ABSTRACT OF THE DISCLOSURE

Data presented herein provide a molecular mechanism for circadian gene *mPer2* in DNA damage response and tumor suppression *in vivo*. Mice deficient in *mPer2* gene display neoplastic phenotypes. These mice are deficient in p53-mediated apoptosis in thymocytes and have increased tumor occurrences after γ-radiation. Core circadian genes are induced by γ-radiation in wild-type mice but not in *mPer2* mutant mice. Temporal expression of genes involved in cell cycle regulation and tumor suppression, such as c-*Myc, Cyclin D1, Cyclin A, Mdm-2* and *Gadd45α* is dependent on mPER2 *in vivo*.